

Pathophysiology Increased Intracranial Pressure (ICP)

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Intracranial pressure (ICP): Alexander Monro observed in 1783 that the cranium is a 'rigid box' containing a 'nearly incompressible brain'. Therefore, any expansion in the contents, especially hematoma and brain swelling, may be initially accommodated by the rejection of fluid components, venous blood and cerebrospinal fluid (CSF). Further expansion is associated with an exponential rise in intracranial pressure. The result is **hypo perfusion and herniation**.

Cerebral blood flow: The brain is dependent on continuous cerebral blood flow for oxygen and glucose delivery, and hence survival. Normal cerebral blood flow (CBF) is about 55 mL/minute for every 100 g of brain tissue. Ischemia results when this rate drops below 20 mL/min, and even lower levels will result in infarction unless promptly corrected. The flow rate is related to **cerebral perfusion pressure (CPP)**, the difference between mean arterial pressure (MAP) and intracranial pressure (ICP):

$CPP (75-105 \text{ mmHg}) = MAP (90-110 \text{ mmHg}) - ICP (5-15 \text{ mmHg})$.

- Risk factor → ↑intracranial volume of CSF → ↑ICP → ↑cerebral perfusion, ↑ brain swelling, cerebral edema → a shift in brain tissue through the dura → herniation → death.
- Increased ICP also leads to brain tissue ischemia/ infarction and brain death.
- Herniation results in a downward shifting of brain tissue from an area of high pressure to low pressure, usually into the brainstem → coma and death.
- Neurosurgical emergencies, especially head injuries, lead to brain swelling, bleeding and hydrocephalus.

- The common pathophysiological pathway is then elevated ICP and reduced CPP and (Cerebral Blood Flow) CBF all this will lead to **ischemia**.
- **Herniation syndromes:** The rapid increase in intracranial pressure which accompanies the exhaustion of compensation mechanisms ultimately results in herniation of brain tissue.
- **Herniation** usually occurring first on the side of any expanding hematoma.
- **Cerebellar herniation** through the foramen magnum compresses medullary Vasomotor and respiratory centers, classically producing **Cushing's triad** of
 - a. hypertension,
 - b. bradycardia and
 - c. irregular respiration.

Causes that can lead to increased ICP:

- a) brain injury, which is often the result of a blow to the head
- b) hydrocephalus, or too much cerebrospinal fluid on the brain
- c) brain swelling
- d) bleeding or blood pooling in the brain
- e) brain aneurysm
- f) brain infection, such as meningitis or encephalitis
- g) stroke
- h) high blood pressure
- i) brain tumour
- j) drug interaction
- k) seizure & epilepsy
- l) hypoxemia, a blood oxygen deficiency

Clinical Presentation

- Slow, bounding pulse and irregular respirations.

- Widening pulse pressure, especially with bradycardia, is highly indicative of elevated ICP.
 - Decreased level of consciousness (**LOC**) with increasing ICP: headache and changes in LOC, slow speech, restlessness, anxiety, confusion, ↑ drowsiness, and inability to be aroused.
 - Stupor, coma, decortication, decerebration, and flaccidity.
 - Sluggishly reactive pupils → fixed and dilated (“blown pupils”). Maybe unequal.
 - Blurred vision, and photophobia.
 - Vomiting.
 - Respiratory impairment (Cheyne-Stokes), irregular or absence of breathing → death.
 - Cushing’s triad or response/reflex: ↓↓ cerebral blood flow → cerebral ischemia → ↑arterial pressure and ↑ SBP, Bradypnoea, widening pulse pressure and reflex bradycardia (late sign).

Complications

- Brainstem herniation, brain anoxia, death
- Diabetes insipidus

Diagnostic Tests

- Serum electrolytes and serum osmolarity
- Cerebral angiography, CT scan, MRI.
- Transcranial doppler (TCD) studies
- **Avoid lumbar** puncture; can lead to brain herniation
- ICP monitoring devices: ventricular drainage, intracranial bolts, intraparenchymal fiberoptic catheter

Management

- Treatment is based on trends and sustained elevations of ICP and low CPP.

The goal is to maintain MAP >90 mm Hg and CPP >70 mm Hg.

■ Administer osmotic diuretic (mannitol [Osmitrol] 0.25–1 g/kg).

Restrict fluids if necessary.

Summary Intracranial pressure

- ❖ Perfusion of the brain with oxygenated blood is critical for its survival
- ❖ Cerebral perfusion pressure is the difference between mean arterial pressure and intracranial pressure
- ❖ Cerebral perfusion is kept constant across a range of perfusion pressures by the process of autoregulation
- ❖ Autoregulation is compromised in the injured brain
- ❖ Increased ICP is an increase in pressure on the brain within the cranium or skull caused by an increase in cerebrospinal fluid (CSF) pressure.
- ❖ Normal ICP is 1–10 mm Hg with an upper limit of 15 mm Hg.
- ❖ Sustained ICP >60 mm Hg is usually fatal.
- ❖ Cerebral perfusion pressure (CPP) is a function of the mean arterial pressure (MAP) and ICP.
- ❖ If the CPP drops to <80 mm Hg, ischemia may occur.
- ❖ A minimum of 50 mm Hg is needed for brain perfusion;
- ❖ brain death occurs if perfusion pressure <30 mm Hg.
- ❖ CPP should be maintained at 70–90 or 80–100 mm Hg and the ICP at <15 mm Hg.
- ❖ $CPP = MAP - ICP$.
- ❖ $MAP = \text{systolic blood pressure (SBP)} + 2 (\text{diastolic blood pressure [DBP]}) \div 3$.

